Efficiency of healing of trophic ulcers when using tissue engineering construction in experiment

Ortiqboyev Farhod Dilshod o'g'li

Assistant of the Department of General Surgery 2 Tashkent Medical Academy

Annotation. Chronic trophic ulcers of the foot and the tibia affect up to 2% of the working population of industrially developed countries. In our scientific work we experimentally examined the effectiveness of wound healing with the use of modern tissue engineering construction collagen enriched with fibroblasts in modeled trophic ulcer in rats of the genus Wistar.

Keywords: Collagen, Trophic Ulcer, Fibroblast, Tissue Engineering Construction

Relevance. The leading cause of long-lasting untenable injuries in the lower extremities is chronic venous insufficiency [9, 11, 12]. Varicose trophic ulcers occur against the background of failure of valves of superficial or perforating veins or after thrombosis of deep veins [6, 7, 10, 13]. The pathological mechanisms of development of trophic disorders in chronic venous insufficiency are common and do not depend on the causes of its occurrence. The pathogenesis is based on macrosomic and microgenomic disorders; even in a horizontal position, the venous system of the extremities increases its capacity by increasing initial tensile strength of the walls of the venous vessels [4].

During the wound process, three phases are distinguished: inflammation, swelling and contracture. Trauma is accompanied by destruction of the epithelial cover, extracellular matrix, endothelium of blood vessels, as a result of which the process of thrombosis is initiated. In addition to the hemostatic function, the blood clot provides the migration of cells into the damage area. The first polymorphic nuclear leukocytes appear in the wound within minutes of injury, reaching a maximum after 24-48 hours, then their number gradually decreases, but even at late stages of healing they are found in inflammatory infiltration. Leukocytes activate the complement system, interact with the calliccrein-kinin system, coagulation and fibriniolisis systems, Hageman factor, arachidonic acid derivatives. With the help of neutrophils and proteolytic enzymes, a partial clot of blood, tissue detritus, foreign bodies, bacterial microflora occurs [3, 5, 15]. As a result of the degradation of platelets, one of the main regulators of the compensatory processes is released - the growth transforming factor (TFP- β), which enhances the expression of the receptor gene in target cells and affects their production of cytokines of growth factors - interleukin-1 (IL-1), as well as the tumor necrosis factor (FNO-a), fibroblast growth factor (FRF), epidermal growth factor (EFG), and thrombotic growth factor (PDGF), the latter increases fibroblast effusion. TFP- β is a powerful chemoattractant for monocytes/macrophages, which are the main source of anti-inflammatory cytokines and fibrous growth factors [1, 2, 9].

Monocytes and macrophages interact with the intercellular matrix and other cell populations through their secreted mediators. Through intergrine receptors, macrophages bind to extracellular matrix (SCM) components, activating phagocytosis. Macrophages stimulate synthesis by mononuclear leukocytes FNO- α (powerful inflammatory factor) and colonic stimulating factor 1 (necessary to maintain a constant number of cells in the damage zone), PDGF, TFP- β , IL-1, FFF, EFD. Infiltration of the damage zone by monocytes/macrophages causes additional TFP- β production, accumulation of WCM, migration and proliferation of fibroblast. TFP- β in parallel blocks the degradation process of WCM [1, 2, 8, 14].

Materials and methods. In order to study and compare the structural-functional changes in wounds with the use of fibroblasts enriched collagen, experimental studies were conducted on the basis of MNIH at the Tashkent Medical Academy.

The study was conducted on 63 white male Wistar rats aged 10 months and weighing 200 20 g. Animals were kept in standard living conditions in individual cells. Animals are divided into three groups of 21 animals in each: control and two experienced.

In the first experimental group, animals with chronic wounds were included, in which regeneration processes were stimulated by applying collagen to the wound.

2-th experimental group consisted of animals with chronic wounds, for activation of regeneration was applied fibroblast-enriched collagen.

The control group included animals with chronic wounds that were treated with ointment of lamelid. By the first day after plasma administration, a weak periphonic tissue edema was observed in 6 (28.6%) animals of the 1st experimental group and 4 (19%) animals of the 2nd experimental group. In the control group, 2 (9.5%) of the animals showed edema with slight diffuse bleeding (probably related to self-injury). All animal groups have blood and exudate clots (figure 1).

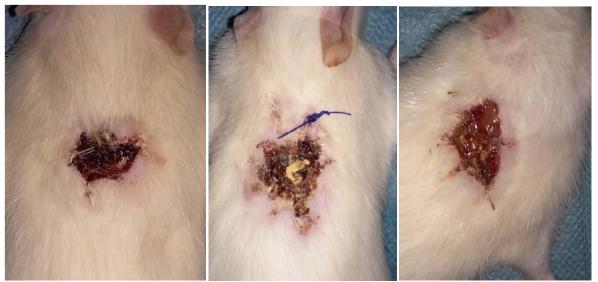


Figure 1 - Wound type in the first day after application of fibroblast-enriched collagen: A 1st Experienced group, B 2nd Experienced group, B - Control group

At 5 days all animals had secondary tension healing, no swelling and hyperemia (figure 2). Planimetric study: average area of wounds in the 1st experimental group - 0.28 cm2, in the 2nd experimental group - 0.38 cm2, in the control - 0.64 cm2.



Figure 2 - Wound type on the 5th day after application of fibroblast-enriched collagen: A 1st Experienced group, B 2nd Experienced group, B - Control group

In the 1st main group (used fibroblasts-enriched collagen) after three days, the area of wounds decreased on average by 37%, by The 5-day treatment was started by 75%. When using collagen (2nd experimental group) after three days, the wound area decreased by 44% of the original one, after five days - by 66%. In the control group at the third day, the wound area decreased by 17%, and on the 5th day - by 43%.

Conclusion. At 5 days from treatment, the greatest reduction in wound area was observed when using collagen-enriched fibroblasts - 9% more than when using collagen and 32% more than in the control group (Figure 2). The experimental data obtained indicate that plasma platelets are highly effective in stimulating regeneration of chronic wounds.

Literature

1. Абаев, Ю. К. Биология заживления острой и хронической раны / Ю. К. Абаев. –// Медицинские новости. – 2003. – № 6. – С. 3–10.

2. Абаев, Ю. К. Заживление острых и хронических ран. Сообщение II / Ю. К. Абаев. // Военная медицина. – 2010. – № 2. – С. 106–110.

3. Альперин, Д. Е. Воспаление: вопросы патогенеза / Д. Е. Альперин. – Москва : Медгиз, 2009. – 146 с.

4. И. М. Васильев, А. В. Муранова, Е. С. Смирнова, Л. И. Богданец [и др.]. / Аспекты патогенеза венозных трофических язв и пути коррекции иммунных нарушений // Клиническая медицина. – 2016. – Т. 94, № 11. – С. 820–826.

5. Болдырев, А. А. Е. И. Кяйвяряйнен, В. А. Илюха. / Биомембранология : учеб. пособие для студентов высш. учеб. заведений, специализирующихся в области биологии, медицины и психологии // Петрозаводск : Карел. науч. центр РАН, 2006. – 225 с.

6. Гафуров, Б. Т. (2023). ПЕРСПЕКТИВЫ И НЕДОСТАТКИ ПРИМЕНЕНИЕ КОЛЛАГЕНА И ДРУГИХ БИОТЕХНОЛОГИЙ В ЛЕЧЕНИЕ ОЖОГОВЫХ РАН (ОБЗОР ЛИТЕРАТУРЫ). European Journal of Interdisciplinary Research and Development, 21, 125-135.

7. Кириенко, А. И. Амбулаторная ангиология / А. И. Кириенко, В. М. Кошкина, В. Ю. Богачев. – Москва : Литтерра, 2007. – 328 с.

8. Е. А. Варюшина, М. А. Анциферова, Г. В. Александров, Т. А. Сазонова [и др.]. / Регуляторная роль интерлейкина-1 при местном воспалении и регенерации тканей в модели кожной раны // Российский аллергологический журнал. – 2012. – № 6. – С. 62–63.

9. Шевченко, Ю. Л. Основы клинической флебологии / Ю. Л. Шевченко, Ю. М. Стойко. – Москва : Шико, 2013. – 336 с.

10. M. E. Vuylsteke, S. Thomis, G. Guillaume, M. L. Modliszewski [et al.]. / Epidemiological Study on Chronic Venous Disease in Belgium and Luxembourg: Prevalence, Risk Factors, and Symptomatology // Europ. J. of Vascular and Endovascular Surgery. – 2015. – Vol. 49, № 4. –P. 432–439. – DOI 10.1016/j. ejvs. 2014.12.031.

11. E. Rabe, J. J. Guex, A. Puskas, A. Scuderi [et al.]. / Epidemiology of chronic venous disorders in geographically diverse populations: results from the Vein Consult Program // Internat. J. of Angiology. -2012. - Vol. 31, No 2. - P. 105-115.

12. C. N. Etufugh, T. J. Phillips. / Etufugh, C. N. Venous ulcers // Clin. Dermatol. – 2007. – Vol. 25, № 1. – P. 121–130.

13. Tohirovich, G. B. (2023). Prospects And Disadvantages the Use of Collagen and Other Biotechnologies in The Treatment of Burn Wounds (Literature Review). Texas Journal of Medical Science, 26, 124-131.

14. Tejero-Trujeque, R. / How do fibroblast interact with the extracellular matrix in wound contraction? // J. Wound Care. -2001. - Vol. 10, No 6. -P. 237-242.

15. Ortiqboyev, F. (2023). TO'QIMA MUHANDISLIK KONSTRUKTSIYALARI, TERI EKVIVALENTLARI VA ULARNI TROFIK YARA KASALIGINI DAVOLASHDA FOYDALANISH. Евразийский журнал медицинских и естественных наук, 3(8), 43-52.